The Epidemiology of ADHD:

Prevalence, Natural History & Clues to Etiology

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Outline

- Prevalence of ADHD and how it varies
- Natural History of ADHD and its impact
- Etiology of ADHD
 - Can exposures to environmental toxicants cause ADHD?
 - How likely is a gene-environment interaction as an explanation?

Estimates of Prevalence of ADHD

Prevalence stated in DSM-IV, TR 3-7 %

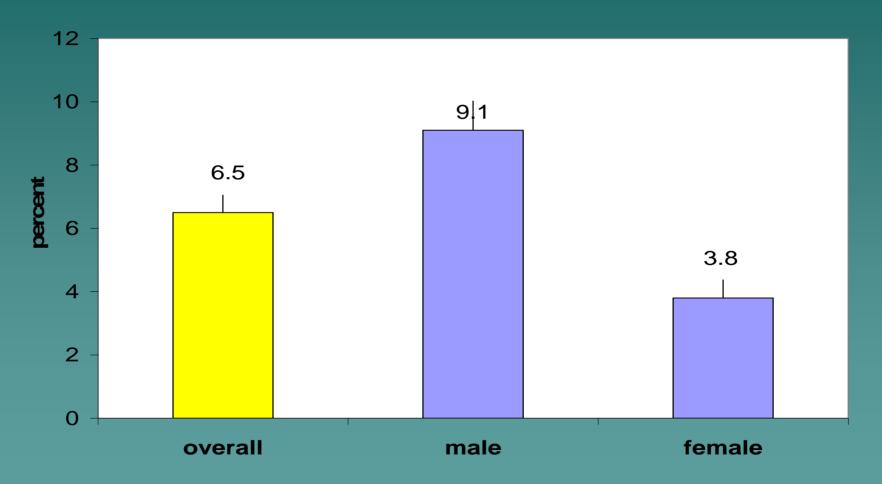
- Many problems with many current estimates: (Skounti et al 2007)
 - Different definitions of ADHD
 - Clinic samples
 - Use of only 1 informant
 - Children taking medication
 - Symptoms caused by other disorders

Epidemiologic Compass

Prevalence varies by:

- Gender
- Age
- Race/Ethnicity
- SES
- Over time
- Geographically

Age-adjusted Estimates of Parent-reported ADHD National Health Interview Survey, 2005



Source: NHIS, Series 10, 231, 2005

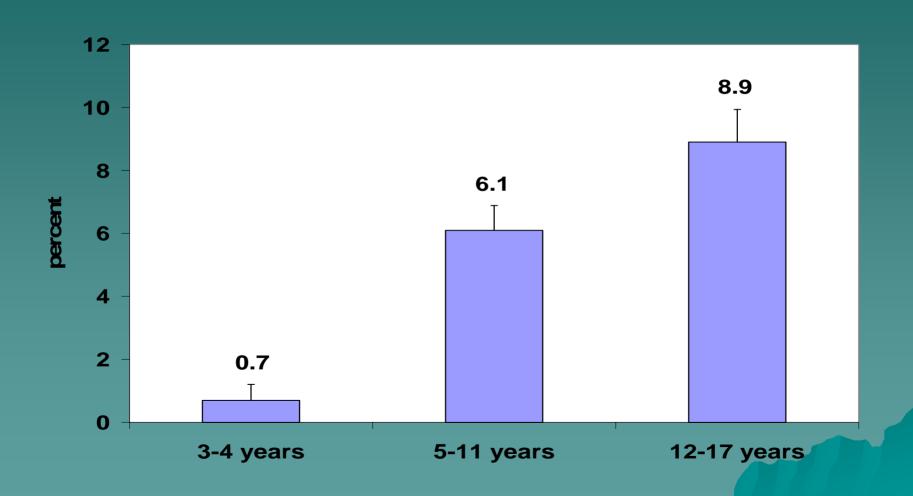
ADHD as a Developmental Disorder

Sex ratio for ADHD is about 3:1

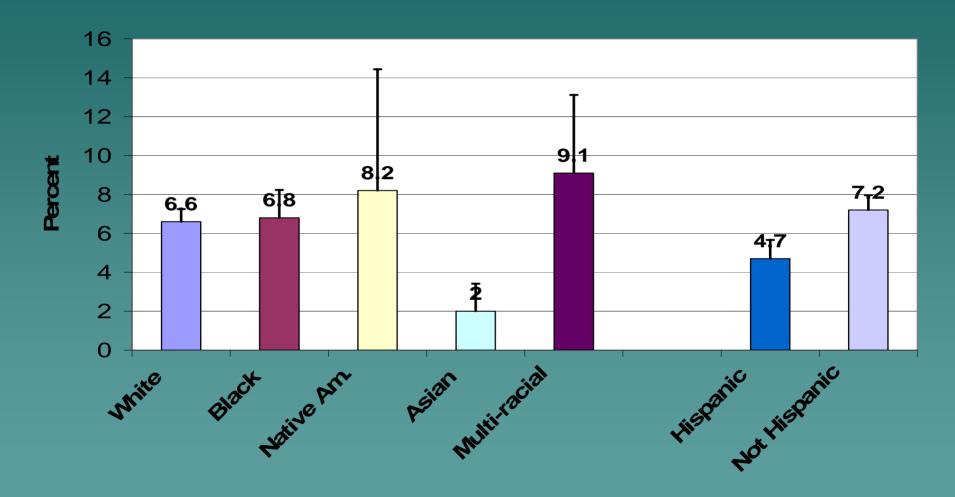
 Male predominance true for many developmental disabilities

•Male vulnerability through birth, infancy, childhood

Relation between Age and Parent-reported ADHD, NHIS, 2005

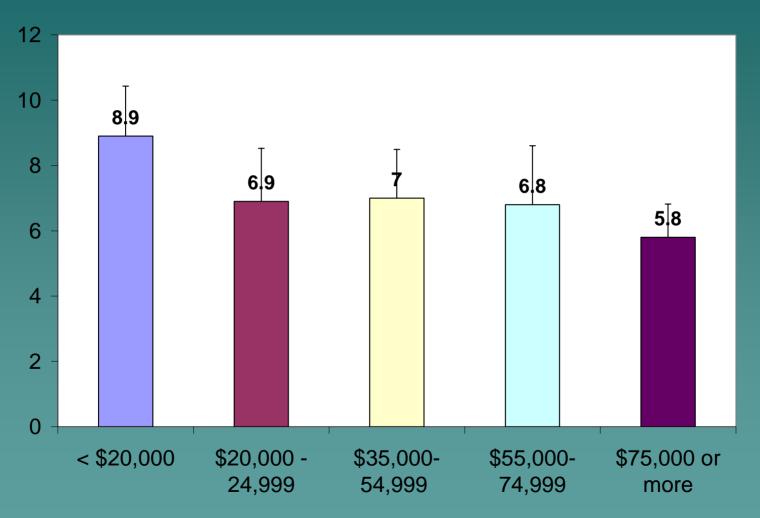


Age-adjusted Prevalence of Parent-Reported ADHD By Race/Ethnicity



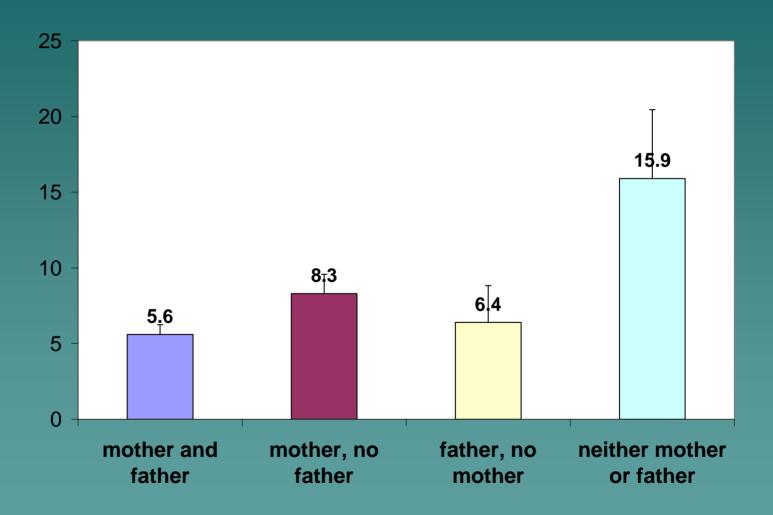
Source: NHIS, 2005

Age-adjusted Prevalence of Parent-reported ADHD by Annual Family Income



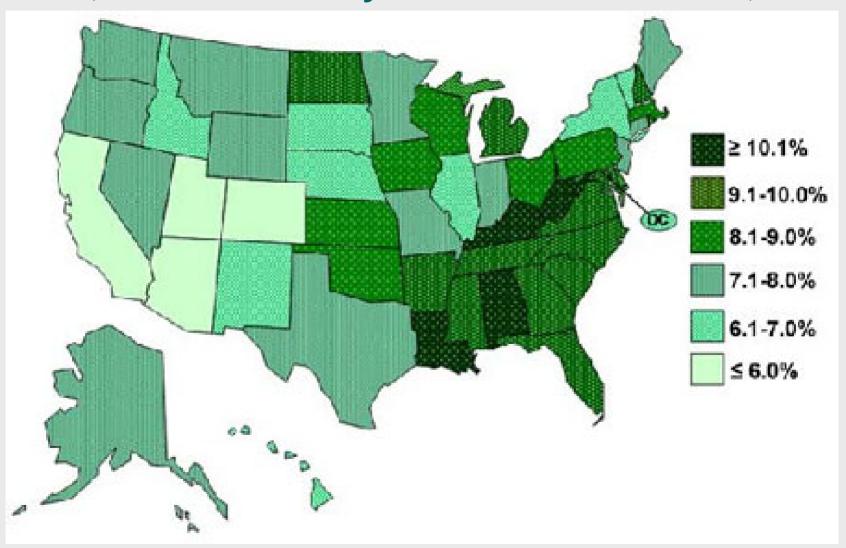
Source: NHIS, 2005

Age-adjusted Prevalence of Parent-reported ADHD By Family Structure



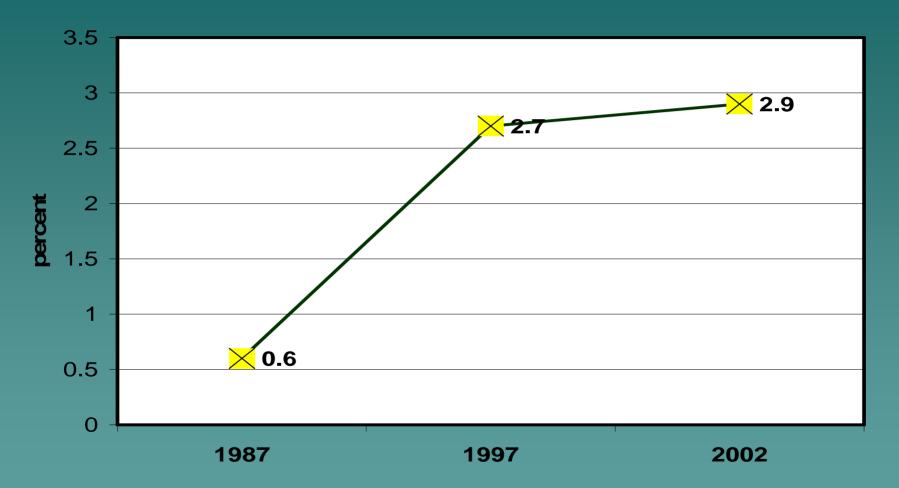
Source: NHIS, 2005

Prevalence of Parent-Reported ADHD by State, National Survey of Children's Health, 2003



National Health

Prevalence of Stimulant Use U.S. Population - Age 18 and Under



Source: Medical Expenditure Database, AHRQ

Impact of Natural History

Percent of ADHD Children with Comordid Conditions Ontario Child Health Study

	% ADHD + Comorbid Conditions	
Males 4-11	53.0	
Females 4-1	42.1	
Males 12-16	36.9	
Females 12-6	67.0	

Source: Szatmari et al. 1989

Natural History of ADHD

Follow-up studies suggest:

- 30-45% will meet criteria for ADHD at age 20
- Risks persist
- Risk of substance abuse and conduction
 disorder

Accidents and Health Risk behaviors

- Long term costs (Discala et al. 1998)
 Accidents
 Health risk behaviors
- Youth with ADHD + Conduct
 Disorder at particularly high risk

Etiology

Genetic Risk of ADHD Farone et al. 2005

- Familial risk
- Heritability estimates
- Many polymorphisms, weak relationships
 May suggest gene-environment interaction

Pregnancy Complications: Collaborative Perinatal Project 1959-1965

Risk of hyperkinetic-impulsive Behavior

- Prenatal smoking
- Hospitalized during pregnancy
- Convulsions during pregnancy
- Breech delivery

Toxicant Exposure and ADHD

- Prenatal exposure to smoking
- Environmental tobacco smoke
- Prenatal exposure to alcohol
- Prenatal stress
- Lead
- Pesticides

Prenatal Smoking and ETS

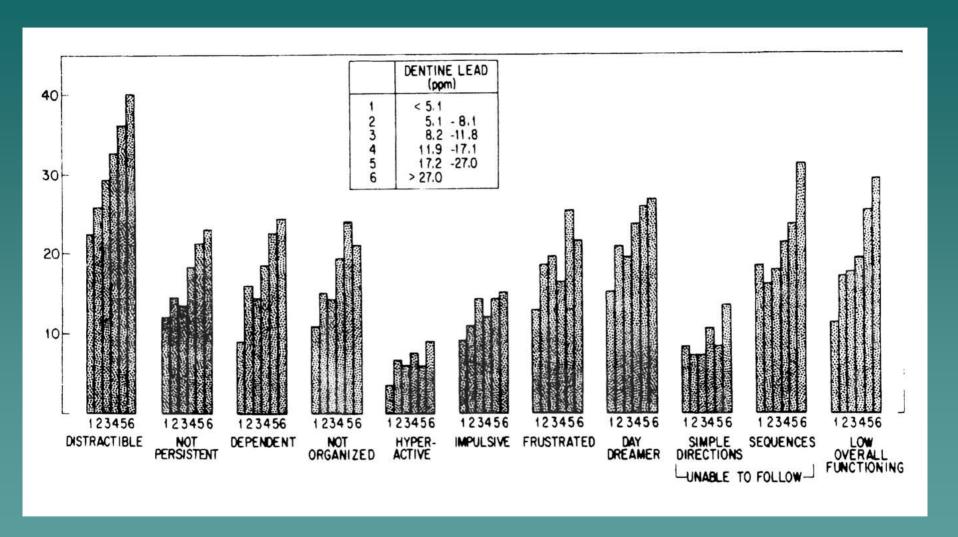
- Prenatal smoking
 - -2003 review (Linnet et al 2003)
 - -evidence mixed, but overall positive

- Environmental tobacco smoe
 - -evidence mixed

Prenatal Alcohol / Prenatal Stress

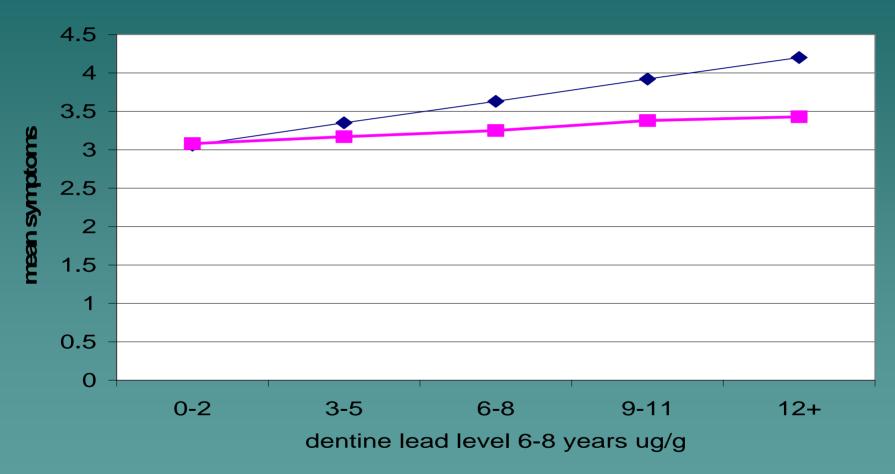
- Nine studies reviewed (Linnet et al, 2003)
 - Evidence mixed
 - Critique comparing ADHD and FAS/FAE
- Implications for environmental studies
- Prenatal stress and ADHD (O'Connor et al 2002, Rodriguez 2005)

Dentine Lead and Teacher-Reported School Problems



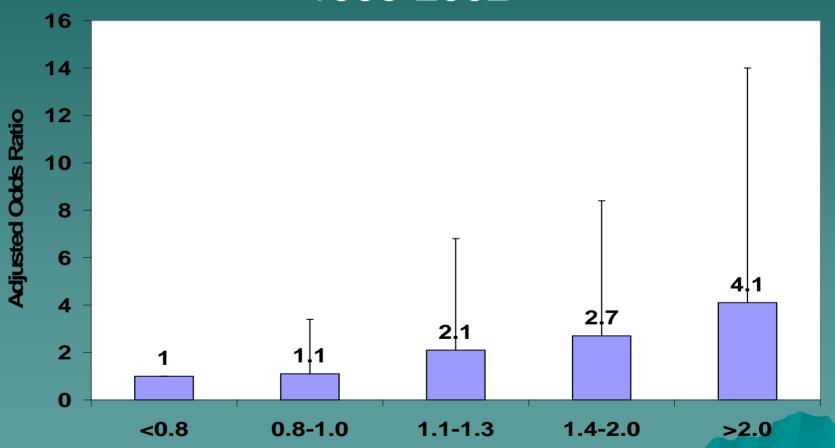
Source: Needleman et al. 1979. N EJ

Relation between Dentine Lead Levels at Age 6-8 and Adjusted Symptoms of Inattention/restlessness at Age 12-13



Source: Fergusson et al. 1993

Relation between Blood Lead and Odds Ratio for ADHD, NHANES 1999-2002



Source: Braun et al. 2006

Adjusted Odds Ratios of Attention Problems at 36 months

Prenatal Exposure	Attention Problems	ADHD Problems
Environmental Tobacco Smoke	2.8 (0.4-17.8)	8.1(1.2-54.7)
Chlorpyrifos	11.3 (1.8-71.0)	6.5 (1.1-38.7)

Source: Rauh et al. 2006

Points to Consider

- Does the endpoint matter?
 - Tests of attention, ADHD symptoms, or ADHD
 - Multi-method approaches
 - Standardization of the case definition
- How can we incorporate social factors into our studies of environmental and genetic risk factors?
- We need to develop more effective ways to control for SES and poverty in our models.

Points to Consider Continued

- Need for more complex models that account for adverse life events and timing of exposures during different stages of child development.
- Epidemiologic tools don't work very well at low exposures. We need to make good use of the tools we do have.

Collaborators

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